

Tuesday, March 5, 1991

Poster Displayed: 2:00PM-5:00PM

Author Present: 2:00PM-3:00PM

Hall F, West Concourse

Arrhythmia—Heart rate Variability: Electrocardiography

Spontaneous variability of ventricular ectopy after myocardial infarction complicates antiarrhythmic therapy control

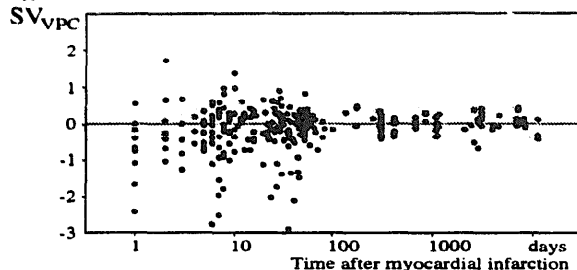
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Spontaneous variability (SV) of single VPCs was determined in 244 patients who survived acute myocardial infarction (AMI). In a subgroup of 30 patients, Holter monitoring was performed continuously over the first 10 days after AMI. In the remaining patients, Holter monitoring was carried out during a chronic state of the disease. SV was assessed by the ratio method developed by our working group previously. The time span between two Holter-ECGs did not exceed one week. Differences in SV were tested for significance by means of the F-test. After AMI, huge spontaneous shifts in the frequency of ventricular arrhythmias were common, as reflected by the extraordinarily high SV of VPCs (see figure). SV dropped to the usual level of a chronic state after two to three months.



We conclude that the high SV of VPCs within the first months after AMI complicates the evaluation of antiarrhythmic drug effects by Holter monitoring during this period. This high SV may reflect the unstable electrophysiological properties of the healing scar.

Long-Term Arrhythmia Monitoring In Healthy, Adult Males

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Through daily Holter (continuous, ambulatory electrocardiographic) monitoring, spontaneous ectopy was assessed over 42-46 sequential days in 37 normal males. This healthy population has been evaluated by history and physical examination, ECG, clinical laboratory tests, exercise testing, and echocardiography; it is representative of the volunteer population of the Lilly Laboratory for Clinical Research. Intersubject ventricular premature complexes (VPCs), expressed as an average daily count, ranged from 0.2 to 287 VPCs/day. The variability in the daily ventricular ectopy was directly proportional to the average daily count (VPCs/day). Daily ectopic counts were not compatible with a Poisson distribution. Elevated levels of ectopic activity (VPCs/day), above baseline, were documented on several, successive days of Holter monitoring in a majority of the subjects. Ectopic (VPCs) periodicity, either within or between days, was not evident. Twenty-four episodes of nonsustained ventricular tachycardia (NSVT) were identified in 16 subjects (43%). NSVT occurred on 1.5% of the monitored days (1,645 Holter days). The number of beats in each NSVT episode ranged from the most frequent count of 3 to an extreme of 35. A positive association was confirmed between the occurrence of NSVT and ventricular pairs.

Intersubject atrial premature complexes (APCs) ranged from 0.3 to 483 mean APCs/day. One hundred (100) episodes of paroxysmal supraventricular tachycardia (PSVT) were documented in 23 volunteers (62%) on 6.1% of the Holter days. A positive association was indicated between the occurrence of PSVT with block and PSVT.

Conclusions:

Ectopy in healthy males exhibits extensive inter- and intrasubject variability, and includes episodes of both NSVT and PSVT in about half of the volunteers, when monitored serially over forty plus days. Temporal patterns do not show periodicity or randomness consistent with a Poisson process.

HEART RATE VARIABILITY IN PATIENTS WITH CONGESTIVE HEART FAILURE AND ISCHEMIC HEART DISEASE BY HOLTER MONITORING
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Analysis of heart rate (HR) variability is useful in assessing the abnormal autonomic control of the heart. It is expected to aid in predicting the sudden cardiac death and the prognosis of patients (pts) with heart disease. Thus we evaluated the autonomic function of the heart by HR variability using 24 hr Holter monitoring. Eighteen pts with congestive heart failure (CHF; NYHA 3,4) and 21 pts with ischemic heart disease (IHD; IVD:5,2-3VD:16) were examined, and were compared with 13 normal control (NL) subjects. HR variability was assessed by the software that determined the RR intervals and standard deviation (SD) of normal sinus beats at successive 5 min intervals over 24 hr period. Six CHF pts were examined both in compensated and in uncompensated stage of heart failure. After Holter monitoring, 6 IHD pts died within 4 weeks. Two pts died in sudden cardiac event. The mean SD in dead IHD pts was significantly lower than that in alive (48±11msec vs 23±7msec p<0.05). Five CHF pts died. The mean SD in dead CHF pts was lower than that in alive (40±15 msec vs 17±6msec p<0.05). The mean SD in uncompensated failure was significantly lower than that in compensated failure (43±12msec vs 30±9msec p<0.05). The mean SD in NL subjects was 57±14msec. These results suggest that the evaluation of HR variability using Holter monitoring is the useful procedure to assess the autonomic severity of CHF and IHD, and to predict the cardiac events.

QRS VOLTAGE-DURATION PRODUCT: ELECTROCARDIOGRAPHIC DETECTION OF LEFT VENTRICULAR HYPERTROPHY

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We examined the hypothesis that QRS duration multiplied by voltage, as an approximation of QRS area, can improve the ECG identification of left ventricular hypertrophy (LVH) in 220 autopsied patients (119 men and 101 women, mean age 60). There were 95 patients with LVH, defined by LV mass >118 g/m² in men and >104 g/m² in women. For each patient, we measured Sokolow-Lyon (SL) voltage, Cornell voltage (RaVL+SV3), and QRS duration, and calculated the product of Cornell voltage and QRS duration (Cornell product) as well as the multivariate Cornell score based on regression of age, sex, P terminal force, Cornell voltage, QRS duration, and T wave amplitude. Receiver operating characteristic (ROC) curves were plotted for each set of criteria and areas under the curves were used to assess overall performance of each test. Sensitivities of each test were compared at matched specificities of 96% by the McNemar modification of chi-square. In this population, the Cornell product performed significantly better (ROC area 0.82) than Cornell voltage (area 0.77, p<0.001) or SL voltage (area 0.71, p<0.005) alone and performed similarly to the multivariate Cornell score (area 0.83, p=ns). At matched specificity of 96%, the Cornell product was significantly more sensitive (44%) than Cornell voltage (33%, p<0.025), SL voltage (23%, p<0.005), or QRS duration alone (23%, p<0.005) for detection of LVH and compared favorably with the sensitivity of the complex Cornell score (40%, p=ns). We conclude that the simple product of Cornell voltage and QRS duration can identify LVH more accurately than voltage criteria alone and may approach or exceed the performance of more complex regression analyses.